THE TRANSMISSION OF RHUS POISON FROM PLANT TO PERSON[1]

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The progress of our knowledge of the transmission of Rhus poison from plant to person reflects, in a general way, the development of our understanding of plants and plant products. This is shown prominently in tracing the history of experiments in regard to the volatility and chemical nature of the poison. In this connection it may be well to consider, besides the dermatitant from *Rhus diversiloba* T. & G., the similar irritant substances from *R. Toxicodendron* L. and from its other sub-species *R. radicans* L.

The earliest explanation of Rhus poisoning attempted was that the plant gives off an invisible colorless vapor, or emanation, which, when breathed or permitted to touch the skin, causes dermatitis. The North American Indian and negro shared in this belief (Thompson, 44).

Some early writers associated Rhus poisoning with the fabulous stories told of the effects of the deadly upas tree (*Ipo toxicaria* Pers., *Antiaris toxicaris* Lesch.) of Java (Bennett, 4).[2]

The theory that the poison is non-volatile has also had its adherents.

[1] The substance of this paper was presented before the Graduate Botanical Club of the University of Pennsylvania, May 6, 1918.

[2] More light on the early physical and chemical nature of the principal irritant poison of this plant may be obtained through a study of the writings of Monti, Hunold, Gleditisch, Achard, Willemet, Pornai, and Krüger. All of these investigators considered the poison volatile. That this conclusion should be drawn at that time is not so remarkable if we consider that the gaseous exchange in plants was not understood at that time. Although Priestly (39) found in 1772 that plants give off oxygen, subsequent repetition of his experiment did not always give the same result. Ingenhousz (20) showed that the air was purified by plants in sunlight. He concluded, however, that the atmosphere is made injurious to animals by emanations from all plants in the shade and at night. Not only were all plants supposed to give off volatile poisons in the shade or at night, but Conradi, Ackerman, and Krauss believed the chief cause of various infectious diseases to be gaseous. As a result we have to this day the word malaria.

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In 1788 Du Fresnoy, experimenting with *R. radicans*, steam-distilled its flowers and leaves. The distillate was not poisonous, but the residue in the still remained toxic.

Fontana (13) experimented with *R. Toxicodendron*. Because of his marked susceptibility to the poison he was forced to stop before he had determined whether or not the poison is volatile.

Two years later, Van Mons (45) collected about fifteen cubic inches of gas given off by a plant of *R. radicans*. Chemical experiments were carried on with this. He then engaged his brother, who was very sensitive to the poison, to hold his hand for more than one hour under a glass bell jar containing gas from the plant obtained in the middle of the day. A month later, not having noticed any eczematous symptoms, he repeated the same experiment with gas collected under a cylinder covered with black cardboard. He felt, even during the immersion, a burning sensation, and developed a typical case of Rhus dermatitis. Van Mons concluded that the poisonous principle of *R. radicans* is a gaseous hydrocarbon which emanates from the plant only at night, on cloudy days, or in the shade.

In 1798, Horsfield, a medical student at the University of Pennsylvania, stated that some people were affected by the exhalations of *R. Vernix* and *R. radicans* to a distance of twenty feet from the plant. He also noticed that dermatitis was produced by the immediate application of the juice of the plant to the external surface of the skin. In analyzing *R. radicans* he placed two pounds of the flowers and leaves with several quarts of water in a small copper still. The distillate was not poisonous, but the residue in the still retained its toxicity.

Lavini (25) considered the poison of *R. Toxicodendron* a gum resin, mixed with a "subtil" acid principle, qualified to combine with the hydrocarbon gas which emanates from the plant after sunset. According to him, the effect of the sap squeezed from the leaves is analogous, but less intense. The effect of "water" distilled from this plant was still less intense.

Khittel (22) attempted a more thorough chemical analysis of *R. Toxicodendron*. Because of his inability to find the poison by the processes outlined, he considered it a volatile alkaloid.

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Millon (35), evidently unaware of the work of Khittel, also investigated *R. Toxicodendron*. He believed the poison a non-volatile gum resin requiring direct contact to cause dermatitis. He found its alcoholic solution to be toxic.

Discussing the experiments of Khittel, Maisch (33) held the poison to be volatile and said:

> It is natural to suppose that, during the process of drying, the greatest portion of the poisonous principle should be lost. This must be still greater, if the dried leaves are powdered, a hot infusion prepared from them, and this infusion evaporated down to the original weight of the dried leaves. It is obvious that Khittel could not have selected a better method for obtaining the least possible quantity of the poisonous principle, if, indeed, it could be obtained by this process at all.

Later Maisch (34) disagreed with Khittel and denied the presence of a volatile alkaloid. He thought that he had found a new volatile acid, which he held to be the active principle and which he called "toxicodendric acid." Maisch enclosed in a tin box a lot of freshly collected leaves of poison ivy, and introduced into this box a number of moistened test papers. The next morning he found that the blue litmus paper had been colored strongly red, whereas curcuma and red litmus paper were unaffected. He writes regarding this experiment:

> This single experiment was at once a conclusive proof that the exhalations of these leaves contained a volatile acid, and that the poisonous properties were most likely due to it.

Maisch describes further how he obtained an impure watery solution of his toxicodendric acid by maceration of the leaves, expression and distillation of the expressed liquid. In preparing his acid, he suffered from a copious eruption and the formation of numerous vesicles on the back of his hands, fingers, wrists, and bare arms. He says further:

> Several persons coming into the room while I was engaged with it were more or less poisoned by the vapours diffused in the room, and I even transferred the poisonous effects to some other persons merely by shaking hands with them. The dilute acid, as obtained by me, and stronger solutions of its salts, were applied to several persons, and eruptions were produced in several instances, probably by the former, though not always, which was not likely owing to the dilute state of the acid.

Maisch did not isolate his acid nor any one of its salts; he never had the substance in question chemically pure. He proved only the presence of a volatile acid. He noticed the characteristic eruption on his own skin while working with the poison ivy. Persons coming to the laboratory at this time were often poisoned. He observed also that an eruption sometimes followed the application of the impure solution of this acid to the skin. From these very rudimentary experiments he drew the wholly unwarranted conclusion that his acid must be the active principle.

By far the most valuable work on *Rhus Toxicodendron* is that of Pfaff (37). From a clinical study of Rhus poisoning, Pfaff came to the conclusion that the poison must be a non-volatile skin irritant. The more volatile

the irritant, the quicker is its action on the skin. Formic acid acts very quickly; acetic acid, less volatile than formic, acts more slowly, but still much more quickly than poison ivy, the latent period of which is usually from two to five days. Pfaff thought that the volatile acid obtained by Maisch might have contained some of the poisonous principle as an impurity, but that it could not produce the dermatitis if prepared in a pure state. He therefore prepared a quantity of the acid by distilling the finely divided fresh plant with steam. The yield was increased by acidulating the mixture with sulphuric acid before the distillation. The acid distillate so obtained was freed from a non-poisonous oily substance by shaking the solution with ether. Barium and sodium salts were made by neutralizing the acid and were purified by crystallization. Analysis showed them to be salts of acetic acid, and they gave the characteristic tests for this acid. The "toxicodendric acid" of Maisch was thus shown to be acetic acid, and not therefore the poisonous principle of the plant.

Pfaff obtained the active principle by the process which he outlines. The lead compounds made in different preparations were analyzed and assigned the formula $C_{21}H_{30}O_4Pb$. The oil itself was not analyzed. Pfaff proposed the name toxicodendrol for the oil. He found that it is not volatile, is decomposed by heat, is soluble in alcohol, ether, chloroform, benzene, etc., but insoluble in water. Its effects upon the human skin were studied in many experiments upon himself and others. It was shown that an exceedingly minute quantity of the poison will produce the dermatitis, even 1/1000 milligram applied in olive oil being active. The oil was given internally to rabbits, its effects being most marked on the kidneys.

Acree and Syme (1) found gallic acid, fisetin, rhamnose, and a "poisonous tar, gum, or wax" in the extract prepared by maceration of the leaves and flowers of poison ivy with ether, and subsequent distillation of the solvent. The lead compound of this poisonous substance was found to be soluble in ether. The authors utilized this property to free the poisonous material from admixed non-poisonous substances. Lead compounds were first prepared by precipitating an alcoholic solution (of the ether extract of the drug) with lead acetate. The precipitate was washed with water, partially dried over sulphuric acid, placed in a Soxhlet apparatus, and extracted with ether until the solvent came over colorless. A green solution was obtained which was washed with water and decomposed with hydrogen sulphide. On evaporating the solvent, a black, poisonous "tar or gum" remained. Upon hydrolysis with 2 percent sulphuric acid, this poisonous substance gave fisetin, rhamnose, and gallic acid. The residue in the thimble was decomposed by hydrogen sulphide, shaken with ether, and evaporated. A hard, brittle, yellow, non-poisonous resin was obtained. The authors believe the poisonous principle of poison ivy to be a complex substance of glucosidal nature.

Chyser in 1910 considered the poison of Rhus a toxalbumin formed by

the combination of a liquid acid with albumin. He puts forth the following evidence in support of this conclusion: (1) the small amount of poison (0.000005 g.) necessary to produce itching and burning on the skin; (2) similarly to a toxalbumin, it loses its toxicity by heating to 50° C. on a water bath; likewise at 75° C. and 100° C. The toxicity was tested by rubbing with a probe on the skin of the upper arm. In no case was irritation evident. This evidence is inconclusive of the poison's being a toxalbumin, for: (1) other substances besides toxalbumins are poisonous when in such small amounts; (2) the poison remains toxic if heated on glass in a steam autoclave for one hour under twenty pounds' pressure per square inch (temperature 126.2° C.); and (3) the poison contains no nitrogen.

The work of Acree and Syme is probably erroneous for: (1) all three of the so-called constituents of the poison are found in two non-poisonous species of Rhus; (2) the natural glucoside yielding fisetin, rhamnose, and gallic acid is non-toxic; and (3) there is not sufficient evidence that the poisonous substance which Syme attempted to decompose was not a complex containing a poisonous body and one or more non-toxic glucosides in addition. McNair (30), working with *R. diversiloba*, concluded that the poison of this plant is not a glucoside of rhamnose, fisetin, and gallic acid. A different method was used for extracting the poison, and none of these substances could be obtained on hydrolysis.

The specific cause of skin poisoning from *R. Toxicodendron* L. and its two sub-species, *R. diversiloba* T. & G. and *R. radicans* L., has thus far been ascribed successively to: an emanation of vapor; a hydrocarbon gas; a gum resin, mixed with a "subtil" acid principle, qualified to combine with hydrocarbon gas which emanates from the plant after sunset; a volatile alkaloid; a non-volatile gum resin; a volatile organic acid (toxicodendric acid); an infection by bacteria (*M. toxicatus*, Burrill, 7); a non-volatile oil (toxicodendrol); a glucoside of fisetin, rhamnose, and gallic acid (toxicodendrin); a toxalbumin; and finally to something other than a glucoside of fisetin, rhamnose, and gallic acid.

The Transmission of Rhus diversiloba Poison

My investigation of the transmission of the poison has been carried on from three standpoints; botanical, chemical, and pathological. The following chemical experiments were carried out:

1. One half pound of fresh, finely chopped poison oak leaves were distilled normally at different temperatures up to the point of decomposition of the leaves. As a result, both the distillate and the residue were non-toxic.

2. Another lot of leaves similarly prepared was subjected to steam distillation. The distillate was non-toxic, but the residue in the retort remained toxic.

3. Distillation, either destructive or with ether, when done under reduced pressure, gave non-toxic distillates.

From the results of these distillation experiments it can be safely argued that the poison is non-volatile and that if non-volatile it can not be carried by entrainment with a volatile substance. It has been considered by some as a non-volatile poison carried by a volatile oil.

In the investigation of the smoke of the burning plant (Von Adelung, 46), leaves were placed in a glass combustion tube. The glass tube was then heated until the leaves began to smoke. The smoke was blown against the skin of a susceptible individual. Dermatitis resulted. The experiment was repeated with the addition of cotton plugs in each end of the tube. Dermatitis did not result.

It was thought that perhaps condensation of the irritant might have occurred on the cotton. The experiment was therefore repeated (McNair, 30), glass wool plugs being used instead of cotton. The glass wool was kept at the same temperature as the burning leaves. No dermatitis resulted. It is concluded, therefore, that the non-volatile poison is carried by particles of soot in smoke.

It is also possible to determine the non-volatility of the poison physiologically. A fresh leaf of poison oak was lightly glued to the concave side of a watch glass about six inches in diameter. The watch glass was then taped on the breast of a susceptible person (the concave side inward) and left there for half an hour. No dermitis resulted. The foregoing experiment was repeated, substituting for the leaf a drop of sap. No ill effects resulted.

A drop of sap was now placed on the skin of a susceptible individual, and the area was covered by a watch glass. Dermatitis occurred after a few hours, but only in the area to which the sap was applied. It did not spread. If the poison were volatilized with moderate ease, at ordinary temperatures, it would have caused a general irritation at, as well as around, the area to which it had been applied. Volatile poisons rapidly penetrate into the tissues, and diffuse there with great ease. Such is the case with the various oils of turpentine, many ethereal oils from the vegetable kingdom, and numerous substances belonging to the aliphatic series, *e.g.*, chloroform and ethyl chloride. Petroleum, benzol, and other compounds of the aromatic series cause local irritation in essentially the same way (Schmiedeberg, 41).

In another experiment, sap was placed on the skin of a susceptible person. After dermatitis had occurred, the affected section of the skin was cut out and thin sections were mounted on microscopic slides. These sections showed that the poison had penetrated but slowly in the skin (McNair). If the poison were volatile, penetration would occur more rapidly and diffusion would be greater.

In ordinary cases of Rhus poisoning, dermatitis is not noticed until

about twelve hours or more after exposure. This long period of latency is much against the supposition that the poison is volatile. It would be much easier for a volatile poison to evaporate and diffuse through the atmosphere in twelve hours if it required a dozen hours to penetrate the skin.

From the preceding experiments, it is clear that the poison is non-volatile. But we still have the question to answer as to how poisoning occurs without contact with the plant. This question has been studied by Von Adelung (46), Schwalbe (43), Hubbard (17), Hadden (16), and Frost (15).

Von Adelung considered the pollen to be toxic and disseminated by the wind. As a matter of fact, the pollen may be rubbed on the skin of a susceptible person without ill effects. The skin may even be lacerated. The pollen grains, although small enough to be carried by the wind, have no wing-like projections or tissues which would aid their flight, but on the contrary are covered with a sticky substance which tends to hold them in masses to the flower. Pollination is effected by insects. Similar non-toxic results have been obtained with the pollen of other poisonous species of Rhus; with that of *R. vernicifera* by Inui (21), that of *R. Vernix* by Warren (48), and that of *R. Toxicodendron* by Rost and Gilg (40).

Schwalbe (43) attributed poison transmission to the trichomes of the plant. The trichomes are very minute and are found in abundance on the young stems and on the under surfaces of the leaves. The trichomes were considered to be poisonous and carried by the wind.

In an investigation of this theory, fresh leaves were placed in an alembic, and a current of air was blown through. The outcoming air current was caused to impinge on the skin of a susceptible individual. No dermatitis resulted. The experiment was repeated, except that the outcoming air was caused to bubble for several hours through alcohol in which the poison is soluble. This alcoholic solution was concentrated and found to be non-toxic. In another experiment, the hairy side of an uninjured leaf (previously examined carefully with a hand lens for the absence of droplets of sap) was drawn across the skin with no ill effects. In another test an uninjured leaf was placed in 95 percent alcohol at room temperature for ten minutes. The alcoholic solution was concentrated and found to be non-toxic.

Rost and Gilg (40) carried on experiments with *R. Toxicodendron* to determine if the plant hairs drop off spontaneously, if they can be blown off from cut twigs, and if the poison, as in *Primula obconica*, can be obtained by contact from the under sides of the leaves. Two shells containing glycerine were placed under Rhus plants for two windy days in May. When this liquid was examined microscopically after the experiment, needle-shaped and club-shaped hairs were found. On October 17, 1911, three wide glass dishes containing glycerine water were placed under thickly leaved branches of *R. Toxicodendron*. These were left for four days. A microscopical examination on October 21 showed no hairs in the dishes.

The preparations contained considerable dust. From the results of these experiments, it is evident that the hairs do not drop off to any great extent spontaneously at either the beginning or the end of the vegetative period.

To determine whether or not the trichomes could be forcibly blown off, five experiments were conducted in 1911:

A. At the end of July (Exp. S. 1 and 2);
B. At the end of August (Exp. S. 3);
C. At the end of September (Exp. S. 4);
D. After the middle of October (Exp. S. 5).

A branch was firmly fastened within a rectangular glass case (100 × 75 × 180 cm.) and was exposed to an air current of about 0.3 atmosphere pressure from a distance of approximately 15 cm. so that the leaves moved as if in a storm. The air current, after passing the leaves, struck an inclined glass plate on which were placed glycerine-covered slides. The current then left the case through a funnel closed with cotton. On the bottom of the glass case two more glycerine-covered slides were placed. During experiments the air current was often interrupted, especially at the beginning and towards the end. This was done to secure the strongest possible disturbances of the leaves. Each experiment lasted at least two hours. Freshly cut branches were used. These branches were afterward pressed and stored, for microscopical examination as to the presence of trichomes. Trichomes were found to have been left on the leaves in abundance.

The glycerine-moistened slides were examined under high and low magnifications. At the end of each experiment, preparations of the dried leaves were made in a chloral-hydrate solution to find if hairs still remained.

The branches used were:

In Series 1:		In Series 2:	
1st day	fresh	1st day	fresh
2d day	one day old	2d day	one day old
3d day	two days old	3d day	three days old

A. *Experimental Series 1* (July 26–28, 1911). Herbarium specimens and two microscopical cross sections gave evidence of many hairs.

I. Wednesday, July 26. The experiment lasted 11 hours. During the first hour the position of the branch was changed twice. A microscopical examination of the glycerine-moistened slides on July 27 showed the absence of club-shaped hairs, but the presence of needle-shaped hairs, much dust, and other impurities.

II. Thursday, July 27. The branch used in the foregoing experiment was exposed to the blast again for two hours (from 11:15 A.M. to 1:15 P.M.). When examined microscopically on July 28, the preparations showed that the dried-up branch as well as the fresh one had not given off club-shaped hairs but only needle-shaped hairs.

III. Friday, July 28. The almost entirely dried branch was subjected for the third time to the air blast (from 10:00 A.M. to 12:00 M.). A microscopical examination on the same day (July 28) showed the presence of needle hairs in all preparations, but of only one club-shaped hair.

At the end of experimental series 1, a chloral-hydrate preparation was made of the entirely dried branch. The under side of the leaves, as is the case in the fresh leaf, were covered with many club-shaped and bristle-like hairs.

B. Experimental Series 2 (July 28–31, 1911). In this series of experiments a branch of densely haired *R. Toxicodendron* was used. A part was pressed and a chloral-hydrate preparation made of it. This showed a dense covering of both kinds of hairs.

I. Friday, July 28. The experiment lasted from 12:30 to 2:30 P.M. On July 29, a microscopical examination disclosed a club-shaped hair in each of four preparations; the remainder showed many needle-shaped hairs.

II. Saturday, July 29. The dried branch was blown on for two hours (from 11:00 A.M. to 1:00 P.M.). A microscopical examination followed on Monday, July 30. This disclosed in

Preparation 1: Three club-shaped hairs, very many needle-shaped hairs, many dirt particles, and pollen grains of other plants.
Preparation 2: No club-shaped hairs.
Preparation 3: Four club-shaped hairs, one with a piece of epidermis.
Preparation 4: Two club-shaped hairs.
Preparation 5: Two club-shaped hairs, one containing yellow protoplasm.
Preparation 6: One club-shaped hair.
Preparation 7: Three club-shaped hairs.
Preparation 8: No club-shaped hairs.

III. Monday, July 31. The three-day-old branch was blown on from 11:30 A.M. to 1:30 P.M. A microscopical examination on August 1 showed one club-shaped hair in each of four out of eight preparations. Both the glycerine-covered slides on the bottom of the case were free from club-shaped hairs. The cotton in the funnel contained no club-shaped hairs (the cotton having been soaked in glycerine and the excess pressed out).

At the end of the experiment, a chloral-hydrate preparation was made from the three-day-old, entirely dried branch. Club-shaped hairs were present in abundance on the leaves. The club-shaped hairs could never wound the cuticle.

Three further experiments were made, similarly to the first two, toward the end of August, in the second half of September, and soon after the middle of October, 1911. The results were similar. In the first days none, or at most one or two, club-shaped hairs could be found in 8 to 10 preparations. In the experiments with the twigs dried two or three days, only a few club-shaped hairs were blown loose. In many experiments in which

preparations of hairs were spread on the skin, not the slightest irritation appeared.

The glycerine layer of one or more experiments was applied and dried on the uninjured skin of the under side of Rost's forearm. The results were negative. Rost was susceptible to the resinous sap of the same shrub. It seems evident, therefore, that the trichomes are non-toxic and are not a means of conveyance of the poison from plant to person.

Hubbard (17) and Hadden (16) thought insects might carry Rhus poison from the plant in ways similar to those by which flies carry bacteria from place to place. This method of transmission seems hardly practicable in many cases. It should be borne in mind that the insect could not transmit the poison by coming in contact with the uninjured plant.

Recently, Frost (15) believed the poison to be bacterial. This has been refuted (McNair, 32).

The methods already discussed constitute all that have been suggested for the transmission of Rhus poison to a distance. As none of them prove very serviceable, we still have to consider the question as to how poisoning occurs without contact with the plant.

It has been found in an examination of the sap that: (1) The unelaborated sap of the xylem is non-toxic; (2) the elaborated sap of the phloem is non-toxic; and (3) the resinous sap of the resin canals is poisonous.

A further examination of the plant tissues shows that the xylem, epidermis, and trichomes which do not contain the resin canals are non-toxic.

When the flowers are examined, it is evident that resin canals do not extend more than half-way up the fully matured stamens, and so it would be expected that the pollen would be non-toxic. The flower of the female plant, on the other hand, contains resin canals in the pistil, and an abundance of resin canals surround the ovule. The ovule remains highly toxic until the seed has fully ripened. The poison, therefore, acts as a protection to the immature seed. This plant thus exemplifies the natural law developed by Kipling (23) that the female is more deadly than the male. It has also been shown (McNair) that the maximum number of cases of Rhus dermatitis recorded in the University of California Infirmary occurs previous to the opening of the flowers.

It has long been known that fresh leaves are more likely to produce poisoning than are dry or fallen leaves. This difference in malignancy has been attributed to a poisonous gas given off by the plant.

Van Mons (45) was convinced by the large number of cases among persons of his acquiantance, that the evil effects of Rhus were produced by a gaseous substance which escaped from the living plant, because the dry leaves or fallen leaves never caused trouble.

Professor Asa Gray also held this same opinion in 1872, as the following letter to Dr. J. C. White discloses:

My personal knowledge that Rhus dried specimens are harmless amounts merely to

this: I handle over and over dried specimens with impunity, but am very sensitive to the fresh plant. Then the poison is volatile, as shown by its affecting persons who do not touch it actually; that of the leaves, I should say, must escape and dry out in the drying process, or in the course of time. In a stem it would not volatilize so soon; but I should not expect to be poisoned from any *old* herbarium specimen, either from twigs or leaves.

Likewise, Mackie (28), writing on the value of oak leaves for forage, says:

It would seem that the irritating and poisonous oil of poison oak is volatile at a comparatively low temperature. In gathering the specimens the writer was badly poisoned even though gloves were worn; yet after drying at ordinary room temperature, and the leaves pressed into the mill with bare hands, no poisoning effects followed.

Opposed to these opinions is the experience of Bogue (6) while investigating an herbarium specimen of *R. venenata* which had been deposited in the Ohio State University not less than three years. He was poisoned by the "sawdust" from the stems of the plant which was the result of borings rom a beetle.

It has previously been conclusively shown that the poison is non-volatile, and the decrease in malignancy of the leaves in drying can be attributed only to a loss of fluidity of the sap and to the loss of toxicity of the poison from oxidation (McNair).

In concluding the botanical investigation, it seems evident that the plant is capable of poisoning only when injured in such a manner that the poisonous resinous sap exudes.

Poisoning without contact with the plant may occur by means of smoke from the burning plant or by contact with substances that have the poisonous sap on them, such as gloves (Hunt, 18; Ward, 47; Frost, 14; Kunze, 24); pocket-knife handles, croquet balls, and botanists' collecting cases (Hunt, 18); hands of another (Hunt, 18; White, 49; Planchon, 38; Cantrell, 10; Maisch, 34); clothing (Balch, 2; White, 49; Bibb, 5; Lindley, 27; Cundell-Juler, 11); shoes one year after contact (Balch, 2; Ward, 47); instruments (Planchon, 38); leather hat bands (Leonard, 26); and firewood (Barnes, 3).

Dermatitis caused by other plants is also sometimes attributed to Rhus; *e.g.*, Cypripedium (Hurlbut, 19); eczema and other eruptions may also be confused with that caused by Rhus.

Conclusions

1. The principal dermatitant of *Rhus diversiloba* is not volatile, for:

(*a*) It is not distillable normally by steam or under reduced pressure. It can not be carried by entrainment with a volatile substance.

(*b*) The smoke of the burning plant is not poisonous when filtered through glass wool at a high temperature.

(*c*) Possible emanations from leaves are non-toxic when (1) the leaves are fastened on the concave side of a watch-glass and then to the skin of a susceptible person; and (2) when a current of air is blown over the leaves and caused to bubble through alcohol, the alcohol is non-toxic.

(*d*) Dermatitis occurs only on the area of skin to which the poisonous sap has been applied; a general irritation as by volatile irritants is not produced.

(*e*) It does not diffuse rapidly in the skin, as is shown microscopically in sections of diseased skin.

(*f*) The period of latency is too long.

2. Portions of the plant which do not cause dermatitis are: the pollen, the trichomes, the epidermis, the cork cells, and the xylem.

3. The poison is confined exclusively to the resinous sap.

4. Leaves decrease in malignancy in drying from the loss of fluidity of the sap and from the oxidation of the poison.

5. Poisoning without contact with the plant may occur from the smoke of the burning plant or by contact with substances that have the poisonous sap on them, such as clothing, shoes, cordwood, tools, the hair of animals, etc.

6. Dermatitis caused by other plants is sometimes attributed to Rhus. There is difficulty in distinguishing eczema from Rhus dermatitis.

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